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Health and Environmental Effects Profile for Benzo(ghi)perylene

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16. ABSTRACT

The Health and Environmental Effects Profile for benzo(ghi)perylene was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine a Reference Dose (RfD) or a carcinogenic potency factor for benzo(ghi)perylene.

The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. Existing data are insufficient to determine an RQ value.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR BENZO(GHI)PERYLENE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

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The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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EXECUTIVE SUMMARY

Benzo(ghi)perylene is a pale yellow-green solid at ambient temperatures. It is soluble in 1,4-dioxane, dichloromethane, benzene and acetone (IARC, 1983), but is practically insoluble in water (Pearlman et al., 1984; Yalkowsky and Valvani, 1979). It reacts with bromomaleic anhydride to form brominated compound and coronene dicarboxylic anhydride derivatives (IARC, 1983). This compound is neither commercially produced nor used in the United States (IARC, 1983). It can be isolated from coal tar (IARC, 1983) or from products of hydrogenation of coal (U.S. EPA/NIH, 1986).

If released to the aquatic environment, benzo(ghi)perylene is not expected to hydrolyze, oxidize (by RO_2 radical or $^1O_2$) or volatilize significantly.

A static culture flask-screening biodegradation study has shown that benzo(ghi)perylene can be significantly biodegraded (Fochtman, 1981); however, microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA/NIH, 1986). In the dissolved state in the water column, direct photolysis may be an important transformation process (U.S. EPA/NIH. 1986); however, a significant portion of the benzo-(ghi)perylene present in the aquatic environment is expected to be in an adsorbed state, which may not be susceptible to phototransformation. Adsorption to suspended particulate matters and sediments in water is an important environmental fate process for this compound. The very low water solubility and high log K_{ow} of benzo(ghi)perylene suggest a significant potential for bioaccumulation; however, PAH may not bioconcentrate appre-ciably in organisms such as fish, that have microsomal oxidase, as this

enzyme metabolizes PAH (Santodonato et al., 1981). Therefore, bioaccumulation potential may be dependent on the organism being considered. The uniformity in the concentration of benzo(ghi)perylene in deep sediment cores of remote lakes (Tan and Heit, 1981) indicate that benzo(ghi)perylene is very persistent under anaerobic and dark conditions. If released to the atmosphere, benzo(ghi)perylene will exist almost entirely in the particulate (adsorbed) phase. The rate of photolysis has been found to vary with the adsorbing substrate (Behymer and Hites, 1985); photolysis of benzo(ghi)-perylene adsorbed to fly ash may have some importance, but adsorption to carbon black stabilizes the compound toward potential phototransformation. Benzo(ghi)perylene may be transported over long distances in the atmospheric aerosol. Removal from the air may occur by wet and dry deposition. If released to soil, benzo(ghi)perylene may be susceptible to slow biodegradation under aerobic conditions. Under most conditions, it is not expected to leach or volatilize from soils.

Human exposure to benzo(ghi)perylene occurs primarily through the inhalation of tobacco smoke and polluted air, and by the ingestion of contaminated food and water (IARC, 1983). It occurs ubiquitously in products of incomplete combustion and in coal tar, and is a component of gasoline and diesel engine exhaust; it also occurs naturally in crude oils (IARC, 1983). It has been widely detected in drinking water, surface water, groundwater, rainwater and aquatic sediments (see Tables 3-1 and 3-2), in many foods (Santodonato et al., 1981; Dennis et al., 1983) and in the ambient atmosphere (see Table 3-3). The average dietary intake of benzo(ghi)perylene in England has been estimated to be 0.21 µg/day (Dennis et al., 1983); the average adult intake in the United States from drinking water has been estimated to be 2.0 ng/day, while the inhalation intake has been estimated

to be 10 ng/day. Its presence in food is a result of contamination from a polluted environment and formation during cooking by pyrolysis (Santodonato et al., 1981; Fazio and Howard, 1983). Its concentration in the ambient atmosphere in the United States has apparently been decreasing over the last 30 years. The ambient air concentration of benzo(ghi)perylene is expected to be higher during winter than summer, apparently because of the burning of fossil fuel for heating during winter months (Greenberg et al., 1985).

Pertinent data regarding the toxicity of benzo(ghi)perylene to aquatic organisms could not be located in the available literature as cited in the Appendix. Several studies reported data concerning residues in aquatic organisms. The following data were reported for species that are commonly eaten by humans: mussels from Norway, trace-87 ng/g (Knutzen and Sortland, 1982); clams from Japan, 0.23-0.95 ng/g (Tsuji et al., 1985); lobsters from eastern Canada, 0.19 ng/g before impoundment and 51 ng/g after impoundment (Dunn and Fee, 1979). Obana et al. (1983) reported BCF values of 1000-1400 for short-necked clams, I. japonica, exposed to 0.06-0.13 µg/ℓ for 1-7 days.

Pertinent data regarding the gastrointestinal or pulmonary absorption of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix. Data from other structurally-related PAH suggest, however, that benzo(ghi)perylene is absorbed readily from the gastrointestinal tract (Rees et al., 1971) and lungs (Kotin et al., 1969; Vainio et al., 1976). In general, PAH are highly lipid soluble and can pass across epithelial membranes (U.S. EPA, 1980).

Pertinent data regarding the distribution, metabolism or excretion of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo(ghi)-perylene in beeswax-trioctanoin mixture into groups of 34 or 35 rats produced a low incidence (4/34) of lung squamous cell carcinomas in the high-dose group after lifetime observation (Deutsch-Wenzel et al., 1983). Lung tumors did not occur in untreated or vehicle treated controls, but IARC (1983) indicated that the tumors in the treated group may be attributable to impurities in the test product, which was 98.5% pure.

Tumorigenicity of benzo(ghi)perylene was not demonstrated in mice in skin application studies involving up to 3 times/week treatment for 12 months (Lijinsky and Saffiotti, 1965; Van Duuren et al., 1970; Van Duuren and Goldschmidt, 1976; Muller, 1968; Hoffmann and Wynder, 1966) in skin initiation-promotion studies using croton oil as a promoter (Hoffman and Wynder, 1966) and PMA as a promoter (Van Duuren et al., 1970) and by bimonthly subcutaneous injections for 5 or 6 months (Muller, 1968). Enhancement of the dermal carcinogenicity of benzo(a)pyrene by simultaneous application of benzo(ghi)perylene has been reported in mice (Goldschmidt et al., 1973; Van Duuren et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of benzo(ghi)perylene.

Benzo(ghi)perylene was mutagenic to S. typhimurium strains TA98, TA100, TA1537 and TA1538 (Andrews et al., 1978; Mossanda et al., 1979; Salamone et al., 1979) and TM677 (Kaden et al., 1979) when assayed in the presence of exogenous metabolic activation preparations. Intraperitoneal injection of benzo(ghi)perylene into hamsters on day 10 of gestation did not induce transformations in the embryo cells (Quarles et al., 1979).

Pertinent data regarding toxic, teratogenic or other reproductive effects of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

Data were insufficient to derive an RfD, RQ, q_1^* or F factor. Benzo-(ghi)perylene was placed in CAG Group D, which means that classification for carcinogenicity and hazard ranking for carcinogenicity are not possible.

TABLE OF CONTENTS

		Page
1.	INTRODUCTION	1
1.1.	STRUCTURE AND CAS NUMBER	1
1.2.	PHYSICAL AND CHEMICAL PROPERTIES	1
1.3.	PRODUCTION DATA	2
1.4.	USE DATA	2
1.5.	SUMMARY	2
2.	ENVIRONMENTAL FATE AND TRANSPORT PROCESSES	3
2.1.	WATER	3
2.1.1.	Hydrolysis	3
2.1.2.	Oxidation	3
2.1.3.	Photolysis	3
2.1.4.	Microbial Degradation	4
2.1.5.	Volatilization	4
2.1.6.	Adsorption	5
2.1.7.	Bioconcentration	5
2.1.8.	Persistence	6
2.2.	AIR	6
2.3.	SOIL	7
2.3.1.	Microbial Degradation	7
2.3.2.	Chemical Degradation	7
2.3.3.	Adsorption	8
2.3.4.	Volatilization	8
2.4.	SUMMARY	8
3.	EXPOSURE	10
3.1.	WATER	10
3.2.	FOOD	14
3.3.	INHALATION	15
3.4.	DERMAL	17
3.5.	SUMMARY	17
4.	PHARMACOKINETCS	18
5.	EFFECTS	19
5.1.	CARCINOGENICITY	19
5.2.	MUTAGENICITY	20
5.3.	TERATUGENICITY	24
5.4.	OTHER REPRODUCTIVE EFFECTS	24
5.5.	CHRONIC AND SUBCHRONIC TOXICITY	24
5.6.	OTHER RELEVANT INFORMATION	24
5.7.	SUMMARY	24

TABLE OF CONTENTS (cont.)

	Page
6. AQUATIC TOXICITY	26
6.1. ACUTE	26
6.2. CHRONIC	26
6.3. PLANTS	26
6.4. RESIDUES	26
6.5. SUMMARY	27
7. EXISTING GUIDELINES AND STANDARDS	28
7.1. HUMAN	28
7.2. AQUATIC	29
8. RISK ASSESSMENT	30
9. REPORTABLE QUANTITIES	32
9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY	32
9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY	32
10. REFERENCES	36
APPENDIX: LITERATURE SEARCHED	46

LIST OF TABLES

No.	Title	Page
3-1	Benzo(ghi)perylene Monitoring Data for Various Types of Water	11
3-2	U.S. Sediment Monitoring Data for Benzo(ghi)perylene	13
3-3	U.S. Air Monitoring Data for Benzo(ghi)perylene	16
5-1	Dermal and Injection Carcinogenicity Studies of Benzo(ghi)perylene	21
5-2	Mutagenicity Testing of Benzo(ghi)perylene	23
9-1	Benzo(ghi)perylene: Minimum Effective Dose (MED) and Reportable Quantity (RQ)	33

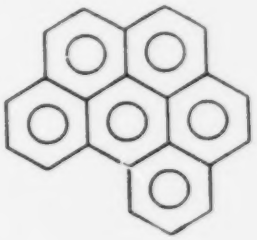
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
K_{oc}	Soil sorption coefficient
K_{ow}	Octanol/water partition coefficient
MED	Minimum effective dose
PAH	Polycyclic aromatic hydrocarbons
PMA	Phorbol myristate acetate
ppb	Parts per billion
ppm	Parts per million
ppt	Parts per thousand
RfD	Reference dose
RQ	Reportable quantity
TLV	Threshold-limit value
TWA	Time-weighted average
UV	Ultraviolet
WS	Water solubility

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Benzo(ghi)perylene is also known as 1,12-benzoperylene (U.S. EPA/NIH, 1986). The structure, empirical formula, molecular weight and CAS Registry number for this chemical are as follows:



Empirical formula: $C_{22}H_{12}$

Molecular weight: 276.3

CAS Registry number: 191-24-2

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Benzo(ghi)perylene is a pale yellow-green crystalline solid at ambient temperature. It is soluble in 1,4-dioxane, dichloromethane, benzene and acetone (IARC, 1983), but is practically insoluble in water. Some of the relevant physical properties of this compound are listed below:

Melting point:	278.3	IARC, 1983
Solubility in distilled water at 25°C:	0.553 µg/ℓ	Pearlman et al., 1984
	0.3 µg/ℓ	Yalkowsky and Valvani, 1979
	0.26 µg/ℓ	Walters and Luthy, 1984
K_{ow}:	7.10 (estimated)	Yalkowsky and Valvani, 1979; Walters and Luthy, 1984
	7.04 (estimated)	Ruepert et al., 1985
Vapor pressure at 25°C:	1.01×10^{-10} mm Hg	Santodonato et al., 1981

Henry's Law constant:	6.6×10^{-8} atm·m³/mol (estimated from water solubility of 0.553 µg/ℓ)	Mabey et al., 1981
	1.44×10^{-7} atm·m³/mol	

Chemically, benzo(ghi)perylene reacts with bromomaleic anhydride to form dibrominated compound and coronene dicarboxylic anhydride derivatives. It also reacts with NO and NO_2 to form nitro derivatives (IARC, 1983).

1.3. PRODUCTION DATA

The compound is neither commercially produced nor imported into the United States (IARC, 1983; USITC, 1984). It can be isolated from coal tar (IARC, 1983) or from products of hydrogenation of coal (U.S. EPA/NIH, 1986).

1.4. USE DATA

There is no known commercial use of this compound (IARC, 1983). Small amounts of benzo(ghi)perylene are used for scientific research.

1.5. SUMMARY

Benzo(ghi)perylene is a pale yellow-green solid at ambient temperatures. It is soluble in 1,4-dioxane, dichloromethane, benzene and acetone (IARC, 1983), but is practically insoluble in water (Pearlman et al., 1984; Yalkowsky and Valvani, 1979). It reacts with bromomaleic anhydride to form brominated compound and coronene dicarboxylic anhydride derivatives (IARC, 1983). This compound is neither commercially produced nor used in the United States (IARC, 1983). It can be isolated from coal tar (IARC, 1983) or from products of hydrogenation of coal (U.S. EPA/NIH, 1986).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

2.1.1. Hydrolysis. Benzo(ghi)perylene contains no hydrolyzable functional groups; therefore, aquatic hydrolysis is not expected to be significant (Mabey et al., 1981).

2.1.2. Oxidation. The rate constants for the oxidation of benzo(ghi)perylene with photochemically produced RO_2 radical and $^1O_2$ have been estimated to be <36 and <360 M^{-1} hr^{-1}, respectively, at 25°C (Mabey et al., 1981). Assuming that concentrations of RO_2 radical and $^1O_2$ in natural water are 10^{-9} and 10^{-12} M (Mabey et al., 1981), respectively, the respective half-lives are estimated to be >2000 and >20,000 years. Therefore, oxidation by RO_2 radical and $^1O_2$ is not environmentally relevant.

2.1.3. Photolysis. In a cyclohexane solvent, benzo(ghi)perylene exhibits UV absorption maxima at 299, 311, 324, 328, 344, 362, 383, 392 and 396 nm (IARC, 1983); therefore, direct photolysis in sunlight is a definite possibility. In the dissolved state in the water column, photolysis may be an important transformation process (U.S. EPA/NIH, 1986); however, a significant portion of the benzo(ghi)perylene in the aquatic environment is expected to be present in the adsorbed state (Section 2.1.6.). Behymer and Hites (1985) examined the atmospheric photolysis of benzo(ghi)perylene adsorbed to various substrates and found relatively significant photolysis when adsorbed to silica gel, alumina or fly ash, but only slow photolysis when adsorbed to carbon black adsorption (Section 2.2.). Therefore, the significance of potential aquatic photolysis may depend on the adsorbing substrate as well as the intensity of the sunlight reaching the water

column. As the depth of water increases, the light intensity will decrease; as a result, photolysis will become increasingly less significant with the increase in the depth of water.

2.1.4. Microbial Degradation. Fochtman (1981) examined the biodegradability of benzo(ghi)perylene in a static culture flask-screening procedure in which 1% of the compound was dissolved in an emulsifier and added to the bacterial suspension (domestic wastewater seed) at a concentration of 1-2 ppm. The suspension was incubated for 7 days. A fresh bacterial suspension was prepared weekly using the seed from the previous week, and the procedure was continued for 28 days. Approximately 60% of the benzo(ghi)perylene was usually degraded during a 7-day incubation. Thus, benzo(ghi)perylene was found to be biodegradable; its biodegradation rate was slightly higher than that of benzo(a)pyrene, which underwent ~41% biodegradation in 7 days.

PAH with at least four aromatic rings are degraded slowly by microbes and biodegradation is considered to be the ultimate fate process in natural water (U.S. EPA/NIH, 1986); however, the concentrations of microorganisms capable of oxidizing the hydrocarbons are extremely low in all but heavily polluted fresh and marine waters, and most species of microorganisms cannot use PAH as a sole carbon source. Microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA/NIH, 1986). Thus, it appears that the significance of biodegradation of benzo(ghi)-perylene in natural waters will depend on the nature of water. In deep, slow moving, turbid water, biodegradation may be more significant than in shallow, rapidly flowing, clear streams.

2.1.5. Volatilization. Using the vapor pressure and water solubility at 25°C, Mabey et al. (1981) estimated the Henry's Law constant for benzo(ghi)-perylene to be 1.44×10^{-7} atm·m³/mol. This value of Henry's Law

constant indicates that the pollutant is essentially nonvolatile (Lyman et al., 1982); therefore, benzo(ghi)perylene is not expected to volatilize significantly from the aquatic environment.

2.1.6. Adsorption. Rump (1984) examined the partitioning of benzo(ghi)-perylene between water and sediment phase with sediments from two rivers. The amount of benzo(ghi)perylene that partitioned to the sediment was found to be 2.5×10^4-3.1×10^5 times greater than the amount that partitioned to the water column.

The estimated K_{oc} values, which are >1 million (Section 2.3.3.), and the widespread detection of benzo(ghi)perylene in various U.S. sediments (Section 3.1.) indicate that adsorption to suspended particulate matter and sediments is an important environmental process. Movement by sediment-sorbed benzo(ghi)perylene is probably an important transport process for this compound (U.S. EPA/NIH, 1986).

2.1.7. Bioconcentration. Estimation of BCF can be made from the following equations (Lyman et al., 1982):

$$\log BCF = 0.76 \log K_{ow} 0.23 \qquad (2\text{-}1)$$

$$\log BCF = 2.791\text{-}0.564 \log (WS \text{ in ppm}) \qquad (2\text{-}2)$$

Based on a water solubility of 0.00026 ppm at 25°C and a log K_{ow} of 7.10 (Walters and Luthy, 1984), the BCF values estimated from Equations 2-1 and 2-2 for benzo(ghi)perylene are 146,500 and 65,000, respectively, which suggest significant bioaccumulation potential. PAH, however, may not appreciably bioconcentrate in organisms such as fish that contain microsomal oxidase, as this enzyme may metabolize PAH (Santodonato et al., 1981). Therefore, the bioaccumulation potential may be very dependent on the organism being considered.

2.1.8. Persistence. Tan and Heit (1981) monitored sediment cores taken from Woods Lake and Sagamore Lake in the remote Adirondack Forest of upstate New York for various PAH. The following benzo(ghi)perylene concentrations (ng/g dry sediment wt) were found at various depths of Woods Lake: 1400 (0-4 cm), 500 (4-8 cm), 180 (8-11 cm), 110 (12-17 cm), 44 (24-26 cm), 46 (42-44 cm) and 59 (80-84 cm). The constancy in the concentration of benzo(ghi)-perylene in the deeper sediment cores indicates that the compound is very persistent under the nonphotolytic, anoxic conditions present in deep sediment cores.

2.2. AIR

Based on a vapor pressure of 10^{-10} mm Hg at 25°C (Mabey et al., 1981), benzo(ghi)perylene is likely to exist almost entirely in the particle-sorbed state in the atmosphere. Yamasaki et al. (1982) monitored the urban air of a Japanese city for an entire year for PAH in both the vapor and particulate phases; the monitoring covered a wide temperature range. The six-ring PAH (including benzo(ghi)perylene, o-phenylenepyrene and anthracene) were detected in the particulate phase but not in the vapor phase, while the three- to five-ring PAH were found in both phases with the partioning ratio depending on temperature.

Behymer and Hites (1985) examined the atmospheric photolysis of benzo-(ghi)perylene adsorbed onto various substrates. With a rotary photoreactor and a medium-pressure mercury lamp as the light source, benzo(ghi)perylene exhibited the following photolytic half-lives in the adsorbed state:

Substrate	Half-Life (hours)
Silica gel	7
Alumina	22
Fly ash	29
Carbon black	>1000

Adsorption to carbon black clearly stabilizes the phototransformation of benzo(ghi)perylene. In the atmosphere, adsorption to stabilizing substrates will allow benzo(ghi)perylene to be transported over long distances in the atmospheric aerosol. Its detection in the remote rural air of South Carolina (Keller and Bidleman, 1984) and in sediments from remote lakes in the Adirondack Forest, NY (Tan and Heit, 1981) may indicate that long-range transport occurs.

Removal of adsorbed benzo(ghi)perylene from the atmosphere may occur by wet and dry deposition. Benzo(ghi)perylene has been detected in rainwater (Lunde et al., 1976; Ligocki et al., 1985). Its presence in lake sediments in the Adirondack Forest, NY, has been attributed to physical deposition (Tan and Heit, 1981).

Since benzo(ghi)perylene does not significantly exist in the vapor phase, atmospheric reaction with photochemically produced HO radical or vapor phase photolysis is not expected to be important.

2.3. SOIL

2.3.1. Microbial Degradation. The soil bacterium Bacillus megaterium has been found to metabolize benzo(ghi)perylene and other PAH (U.S. EPA/NIH, 1986); however, sufficient data are not available to predict the significance of the soil biodegradation for benzo(ghi)perylene. Based on the data in Section 2.1.4., benzo(ghi)perylene may be slowly biodegraded in soil under aerobic conditions.

2.3.2. Chemical Degradation. Pertinent data regarding the chemical transformation of benzo(ghi)perylene in natural soils could not be located in the available literature as cited in the Appendix.

2.3.3. Adsorption. Estimation of K_{oc} can be made from the following regression equations (Lyman et al., 1982):

$$\log K_{oc} = 0.44\text{-}0.54 \log (\text{WS in mol fraction}) \quad (2\text{-}3)$$

$$\log K_{oc} = 1.00 \log K_{ow} \; 0.21 \quad (2\text{-}4)$$

Based on a water solubility of 0.26 µg/ℓ at 25°C and a log K_{ow} of 7.10 (Walter and Luthy, 1984), the K_{oc} values estimated from Equations 2-3 and 2-4 for benzo(ghi)perylene are 1.8 and 7.8 million, respectively, which suggests soil immobility; however, the detection of benzo(ghi)perylene in several groundwaters (Section 3.1.) indicates that leaching can occur under certain conditions. Leaching may occur in soils that contain low organic matter (e.g., sand), and may also occur from sites subjected to spills or disposal of appropriate petroleum and coal materials that contain high concentrations of PAH including benzo(ghi)perylene; however, benzo(ghi)perylene is not expected to leach in soil under most conditions.

2.3.4. Volatilization. Benzo(ghi)perylene cannot be expected to volatilize significantly from soil surfaces because of its very low vapor pressure of 10^{-10} mm Hg at 25°C and a very high K_{oc} value of ~10^6.

2.4. SUMMARY

If released to the aquatic environment, benzo(ghi)perylene is not expected to hydrolyze, oxidize (by RO_2 radical or $^1O_2$) or volatilize significantly. A static culture flask-screening biodegradation study has shown that benzo(ghi)perylene can be significantly biodegraded (Fochtman, 1981); however, microbial oxidation of PAH requires oxygen and will not proceed in anoxic sediments or water (U.S. EPA/NIH, 1986). In the dissolved state in the water column, direct photolysis may be an important transformation process (U.S. EPA/NIH, 1986); however, a significant portion of the benzo(ghi)perylene present in the aquatic environment is expected to be in an adsorbed state, which may not be susceptible to phototransformation.

Adsorption to suspended particulate matters and sediments in water is an important environmental fate process for this compound. The very low water solubility and high log K_{ow} of benzo(ghi)perylene suggest a significant potential for bioaccumulation; however, PAH may not bioconcentrate appreciably in organisms such as fish, that have microsomal oxidase, as this enzyme metabolizes PAH (Santodonato et al., 1981). Therefore, bioaccumulation potential may be dependent on the organism being considered. The uniformity in the concentration of benzo(ghi)perylene in deep sediment cores of remote lakes (Tan and Heit, 1981) indicates that benzo(ghi)perylene is very persistent under anaerobic and dark conditions. If released to the atmosphere, benzo(ghi)perylene will exist almost entirely in the particulate (adsorbed) phase. The rate of photolysis has been found to vary with the adsorbing substrate (Behymer and Hites, 1985); photolysis of benzo(ghi)perylene adsorbed to fly ash may have some importance, but adsorption to carbon black stabilizes the compound toward potential phototransformation. Benzo(ghi)-perylene may be transported over long distances in the atmospheric aerosol. Removal from the air may occur by wet and dry deposition. If released to soil, benzo(ghi)perylene may be susceptible to slow biodegradation under aerobic conditions. Under most conditions, it is not expected to leach or volatilize from soils.

3. EXPOSURE

Human exposure to benzo(ghi)perylene occurs primarily through the inhalation of tobacco smoke and polluted air, and by the ingestion of contaminated food and water (IARC, 1983). It occurs ubiquitously in products of incomplete combustion, in coal tar and in gasoline engine exhaust; it also occurs naturally in crude oils (IARC, 1983).

3.1. WATER

Table 3-1 lists various benzo(ghi)perylene monitoring data for drinking, ground, surface and rain water; Table 3-2 lists sediment monitoring data at various U.S. locations. In an analysis of the U.S. EPA STORET database, benzo(ghi)perylene was positively detected in 1.0% of 914 surface water observation stations and 1.5% of 1211 effluent reporting stations (Staples et al., 1985). Griest (1980) detected benzo(ghi)perylene at concentrations of 7.3 µg/g (dry sediment) in the sediment and 2.0 µg/ℓ in the water in an effluent wastewater channel from a coking plant. Walters and Luthy (1984) detected levels $\leq$90 µg/ℓ in wastewaters from two coal coking plants.

In general, PAH can be released to water from industrial and municipal effluents, atmospheric fallout and precipitation, road runoff (tire wear, bitumen and asphalt surfaces, cracked lubricating oils) and marine shipping and harbor oil (Santodonato et al., 1981). Herrmann and Huebner (1982) have suggested that most PAH pollution of rivers in urban areas originates from storm water overflows containing street and roof dust washed off during storms; the contribution of precipitation-borne PAH may be comparatively small in such instances.

TABLE 3-1

Benzo(ghi)perylene Monitoring Data for Various Types of Water

Type of Water	Concentration (ppt)	Location	Sampling Date	Reference
Drinking (finished)	0.4	Syracuse, NY	1976-1977	Saxena et al., 1977
	0.7	Buffalo, NY		
	0.7	Pittsburg, PA		
	2.5	Huntington, WV		
	2.9	Endicott, NY		
	1.9	Hammondsport, NY		
	4.0	Philadelphia, NY		
	1.8	New York, NY		
	2.6	Lake George, NY		
	2.2	New Orleans, LA		
	<1.0	Cincinnati, OH	NR*	Sorrell et al., 1980
	<1.0	New Orleans, LA		
	<1.0	Miami, OH		
	<1.0	Seattle, WA		
	<1.0	Columbus, OH		
	<1.0	Cape Girardeau, MO		
	<1.0	Wheeling, WV		
Drinking	0.8-7.1	NR	NR	IARC, 1983
Groundwater	0.8-17.5	Germany	1963-1964	Sorrell et al., 1980
	1000 (maximum)	Netherlands	1976-1978	Zoeteman et al., 1981
	0.7-6.4	NR	NR	IARC, 1983
Surface	0.3-28.5	NR	NR	IARC, 1983

TABLE 3-1 (cont.)

Type of Water	Concentration (ppt)	Location	Sampling Date	Reference
Surface (river)	3.2-134.0	Germany	1963-1964	Sorrell et al., 1980
	30-40	England (Thames River)	NR	
	2.0-11.3	Severn River	NR	
	0.4-11.0 (in solution)	River Trent	NR	
	4.5 (maximum)	Germany	NR	Hermann and Heubner, 1982
	19	England	NR	Crane et al., 1980
Surface (lake)	NR	Lakes Erie, Ontario, Michigan and Superior	NR	Great Lakes Water Quality Board, 1983
Rainwater	NR	Norway	1975	Lunde et al., 1976
	6.0 (average)	Portland, OR	1984	Ligocki et al., 1985
	2.3-10.8	NR	NR	IARC, 1983

*Compound was qualitatively detected

NR = Not reported

TABLE 3-2

U.S. Sediment Monitoring Data for Benzo(ghi)perylene

Concentration (ng/g)	Location	Sampling Date	Reference
32-120	Cayuga Lake, NY (deep water)	1978	Heit, 1985
14-130	Cayuga Lake, NY (littoral sediment)		
12-32	Washington State (bottom river sediment)	1979-1980	Prahl et al., 1984
7-150	Columbia River (suspended sediment)		
70-600	Lake Erie	NR	Eadie et al., 1982
1400 (0-4 cm depth) 500 (4-8 cm depth) 59 (80-84 cm depth)	Woods Lake, NY	NR	Tan and Heit, 1981
620	Puget Sound, WA	NR	Varanasi et al., 1985

NR = Not reported

Based on the monitoring data from Table 3-1, the concentration of benzo(ghi)perylene in drinking water may be ~1.0 ppt (ng/ℓ). Assuming an average daily water intake of 2.0 ℓ for an adult, the intake of benzo-(ghi)perylene from drinking water is estimated to be 2.0 ng/day.

3.2. FOOD

In general, PAH found in food are present as a result of contamination from a polluted environment or are formed during the cooking (pyrolysis) process (Santodonato et al., 1981; Fazio and Howard, 1983); minute amounts of this chemical may also originate from geochemical or biosynthetic sources (Fazio and Howard, 1983).

The following benzo(ghi)perylene concentrations (in ppb) have been reported for various foods (Santodonato et al., 1981): corn oil (0.6), sunflower oil (4.0), soybean oil (1.0), olive oil (0.9), peanut oil (0.9), smoked herring (2.4), smoked cod (2.2), smoked mackerel (0.2-0.3), charcoal-broiled steaks (6.7), barbecued ribs (4.7), smoked ham (1.4), barbecued beef (4.3), smoked bologna (0.04-0.20), smoked mortadella (0.4), heavily smoked bacon (3.0), charcoal-broiled hamburger (trace-14.9), broiled T-bone steak (6.2-12.4) and heavily smoked ham (2.5-25.0).

Dennis et al. (1983) examined total-diet samples of food groups in England and found the following mean benzo(ghi)perylene concentrations (in µg/kg) in the various food groups: cereal (0.28), meat (0.05), fish (0.12), oils and fats (1.26), fruit and sugar (0.06), vegetables (0.06-0.08), beverages (0.01) and milk (0.01). Dennis et al. (1983) then estimated the total benzo(ghi)perylene dietary load in England to be 0.21 µg/person/day. Based on monitoring of total-diet samples collected in the Netherlands, Vaessen et al. (1984) estimated the maximum food intake to be 0.9 µg/person/day.

3.3. INHALATION

Table 3-3 lists recent (1979-1983) U.S. ambient air monitoring data for benzo(ghi)perylene; in general, the mean concentrations detected are <2.0 ng/m³. The average U.S. urban atmosphere in the early 1960s has been reported to have had a concentration of ~8 ng/m³ (Santodonato et al., 1981). In 1952-53, the airborne concentration of benzo(ghi)perylene in Los Angeles, CA, was reported to be 21 ng/m³, which was slightly >3 times the concentration in 1958-59 (Gordon and Bryan, 1973). These monitoring data suggest that the ambient atmospheric load of benzo(ghi)perylene has been generally decreasing over the past 30 years. This decrease may be a result of decreases in coal consumption for residential heating and industrial uses, restrictions on open burning, and improved efficiency for stationary incineration and combustion sources with improvements of pollution control. The higher atmospheric levels of benzo(ghi)perylene in wintertime vs. summertime air in New Jersey (see Table 3-3) may be a reflection of an increased use of fossil fuel combustion for heating purposes.

Assuming an average ambient air benzo(ghi)perylene concentration of ~0.5 ng/m³ (from Table 3-3) and a human air intake of 20 m³/day, the average daily intake can be estimated to be 10 ng. Matsumoto and Kashimoto (1985) used Japanese monitoring data to estimate an average daily intake of 27 ng.

In general, PAH are emitted to the atmosphere by the combustion of fossil fuels (oil, coal), by vehicular exhausts, by open burning (agricultural burning, forest fires, structural fires, refuse burning) and by the burning of wood, especially for residential heating (NRC, 1983).

Benzo(ghi)perylene has been identified in mainstream cigarette smoke 1-39 ng/cigarette), sidestream cigarette smoke (98 ng/cigarette), smoke-filled rooms (17 ng/m³) and mainstream marijuana smoke (0.7 ng/cigarette) (IARC, 1983).

TABLE 3-3

U.S. Air Monitoring Data for Benzo(ghi)perylene (1979-1983)

Concentration (ng/m^3)	Location	Sampling Date	Reference
0.09-0.56 (mean)	four cities, New Jersey	summer 1981	Harkov et al., 1984
0.09-3	Columbia, SC	1981-1982	Keller and Bidleman, 1984
0.02	rural South Carolina	1982	
0.11-1.0 (average)	three sites, Washington State	1979	Prahl et al., 1984
2.0 (average)	Portland, OR	1984	Ligocki et al., 1985
0.59-2.74 (mean)	four cities, New Jersey	winter 1982	Greenberg et al., 1985
0.02-0.62 (mean)		summer 1982	
0.20-1.31 (mean)		winter 1983	
1.0 (mean)	Waterbury, VT	winter 1982	Sexton et al., 1985

3.4. DERMAL

Pertinent data regarding dermal exposure to benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

3.5. SUMMARY

Human exposure to benzo(ghi)perylene occurs primarily through the inhalation of tobacco smoke and polluted air, and by the ingestion of contaminated food and water (IARC, 1983). It occurs ubiquitously in products of incomplete combustion and in coal tar, and is a component of gasoline and diesel engine exhaust; it also occurs naturally in crude oils (IARC, 1983). It has been widely detected in drinking water, surface water, groundwater, rainwater and aquatic sediments (see Tables 3-1 and 3-2), in many foods (Santodonato et al., 1981; Dennis et al., 1983) and in the ambient atmosphere (see Table 3-3). The average dietary intake of benzo(ghi)perylene in England has been estimated to be 0.21 µg/day (Dennis et al., 1983); the average adult intake in the United States from drinking water has been estimated to be 2.0 ng/day, while the inhalation intake has been estimated to be 10 ng/day. Its presence in food is a result of contamination from a polluted environment and formation during cooking by pyrolysis (Santodonato et al., 1981; Fazio and Howard, 1983). Its concentration in the ambient atmosphere in the United States has apparently been decreasing over the last 30 years. The ambient air concentration of benzo(ghi)perylene is expected to be higher during winter than summer, apparently because of the burning of fossil fuel for heating during winter months (Greenberg et al., 1985).

4. PHARMACOKINETICS

Pertinent data regarding the gastrointestinal or pulmonary absorption of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix. Data from other structurally-related PAH suggest, however, that benzo(ghi)perylene is absorbed readily from the gastrointestinal tract (Rees et al., 1971) and lungs (Kotin et al., 1969; Vainio et al., 1976). In general, PAH are highly lipid soluble and can pass across epithelial membranes (U.S. EPA, 1980).

Pertinent data regarding the distribution, metabolism or excretion of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the tumorigenic potential of orally-administered benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

Mixtures of heated beeswax and trioctanoin (1:1) containing 0.16, 0.83 or 4.15 mg benzo(ghi)perylene (98.5% pure) were injected into the left lung of groups of 34 or 35 female Osborne-Mendel rats after thoracotomy (Deutsch-Wenzel et al., 1983). Additional groups of 35 rats served as vehicle, untreated and positive [0.1, 0.3 or 1.0 mg benzo(a)pyrene] controls. All rats were necropsied at the time of natural death or when sacrificed at moribundity, but histological examinations were limited to the lungs and organs showing gross abnormalities. Median survival times were 109, 114, 106, 104 and 118 weeks in the low-, middle- and high-dose treated groups, the vehicle control and the untreated control groups, respectively. Squamous cell carcinomas of the lung occurred in 0/35, 1/35 and 4/34 mice in the low-, mid- and high-dose treated groups, respectively. Lung tumors were not observed in either the vehicle or untreated control groups, but carcinomas occurred at dose-related incidences in the positive controls (4/35, 21/35, 33/35). The low incidences of lung tumors in the treated mice were considered to indicate a weak effect of benzo(ghi)perylene in this assay, but it was noted that the response might have been caused by impurities in the 98.5% pure product that was tested. The results of this study should therefore be regarded as inconclusive.

Benzo(ghi)perylene has also been tested for tumorigenicity in mice in skin application studies involving up to 3 times/week treatment for 12 months (Lijinsky and Saffiotti, 1965; Van Duuren et al., 1970; Van Duuren and Goldschmidt, 1976; Muller, 1968; Hoffmann and Wynder, 1966); in skin

initiation-promotion studies using croton oil as a promoter (Hoffman and Wynder, 1966) and PMA as a promoter (Van Duuren et al., 1970); and by bimonthly subcutaneous injections for 5 or 6 months (Muller, 1968). The results of these studies were negative (Table 5-1). Although it is difficult to evaluate carcinogenic potential solely on the basis of results from these types of assays, it should be noted that positive responses were obtained in many of the studies with PAH of known carcinogenicity (e.g., benzo(a)pyrene). Enhancement of the dermal carcinogenicity of benzo(a)-pyrene by simultaneous application of benzo(ghi)perylene has been reported in mice (Goldschmidt et al., 1973; Van Duuren et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of benzo(ghi)-perylene.

5.2. MUTAGENICITY

Benzo(ghi)perylene has been reported to produce mutation to histidine independence in Salmonella typhimurium strains TA98, TA100, TA1537 and TA1538 when assayed in the presence, but not the absence, of exogenous metabolic activation preparations (Andrews et al., 1978; Mossanda et al., 1979; Salamone et al., 1979). Benzo(ghi)perylene also produced mutation to 8-azoguanine resistance in S. typhimurium TM677 in the presence of activation preparations (Kaden et al., 1979). These studies are summa-rized in Table 5-2.

Intraperitoneal injection of 30 mg/kg benzo(ghi)perylene into hamsters (strain unspecified) on day 10 of gestation did not induce neoplastic trans-formation in embryo cells that were first cultured on day 13 (Quarles et al., 1979) (see Table 5-2). Additional information regarding the genotoxic potential of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

TABLE 5-1

Dermal and Injection Carcinogenicity Studies of Benzo(ghi)perylene

Route	Species/Strain	No./Sex[a]	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/Swiss	NR/F	NR	saturated (0.38%) solution in benzene; volume applied and treatment schedule not reported	life	No skin tumors; primary report not available	Lijinsky and Saffiotti, 1965
	mouse/a ICR/Ha/Swiss	20/F	purified	single application of 0.8 mg in benzene	58-60 weeks	No skin tumors in treated group or vehicle or untreated controls	Van Duuren et al., 1970
	mouse/NMRI	50/F	NR	0.2 ml of 0.01% solution 0.4 ml of 0.5% solution or 0.8 ml of 0.5% solution 2 times/week for 25 weeks; dichloromethane vehicle; total doses 1, 100 and 200 mg, respectively	675	No skin tumors in any of the vehicle control groups; 64-72% survival in all groups at end of study; 100% skin tumor incidences in mice similarly treated with benzo(a)pyrene; primary report not available	Muller, 1968
	mouse/Ha/ICR/ Mil/Swiss	20/F	chromato-graphically-purified	0.05 or 0.1% in 1,4-dioxane, 3 times/week for 12 months	15 months	Skin tumors in 1/20 (low dose; papillomas) and 0/20 (high dose); none in 20 vehicle controls; high incidences of skin tumors in mice similarly treated with benzo(a)pyrene	Hoffmann and Wynder, 1966
	mouse/ ICR/Ha/Swiss	50/F	recrystal-lized	21 µg in acetone, 3 times/week for 368 days	NR	No skin tumors in treated, vehicle control or untreated control groups	Van Duuren and Goldschmidt, 1976
	mouse/Ha/ICR/ Mil/Swiss	30/F	chromato-graphically-purified	0.025 ml of 0.1% (0.25 mg) in 1,4-dioxane every 3 days for 28 days; application of 2.3 mg croton oil from day 28 but schedule not specified	6 months	Skin papillomas in 2/30 vs. 2/30 in croton oil, control, 0/30 in vehicle control and 24/30 in benzo(a)pyrene control	Hoffman and Wynder, 1966
	mouse/ ICR/Ha/Swiss	20/F	purified	single application of 0.8 mg in benzene followed 2 weeks later by 3 times/week application of PMA for life	360-400 days	Skin papillomas in 3/20 and carcinomas in 1/20 vs. 1/20 with papillomas in PMA controls, 0/20 in vehicle controls and 0/100 in untreated controls	Van Duuren et al., 1970

TABLE 5-1 (cont.)

Route	Species/Strain	No./Sex*	Purity	Treatment	Duration	Effects/Comments	Reference
Skin	mouse/ ICR/Ha/Swiss	50/F	high	applied together with 5 µg benzo(a)pyrene in acetone at molar ratios (benzo(ghi)perylene:benzo-(a)pyrene) of 1:1 or 3:1, 3 times/week for 33 weeks for 33 weeks	33 weeks	Benzo(ghi)perylene enhanced the skin tumorigenicity of benzo(a)-pyrene, particularly at 3:1 ratio; responses not reported; study reported as an abstract	Goldschmidt et al., 1973
	mouse/ ICR/Ha/Swiss	50/F	high	21 µg applied together with 5 µg benzo(a)pyrene in acetone, 3 times/week for 52 weeks	52 weeks	Incidences of skin papillomas and carcinomas were 20/50 and 17/50 in treated vs. 13/50 and 10/50 in group treated with benzo(a)pyrene alone and no skin tumors in vehicle or untreated controls	Van Duuren et al., 1973
	mouse/ ICR/Ha/Swiss	50/F	recrystal-lized	7 or 21 µg applied together with 5 µg benzo(a)pyrene in acetone 3 times/week for 368 days	NR	Incidences of skin papillomas were 19/50 (7 µg) and 20/50 (21 µg) vs. 14/50 in group treated with benzo(a)pyrene alone and 0/50 in group treated with benzo(ghi)perylene alone; similar pattern of squamous carcinomas also induced	Van Duuren and Goldschmidt, 1976
Subcutaneous	mouse/NMRI	50/F	NR	0, 0.83 or 16.7 mg suspended in aqueous gelatin once every 2 weeks for 6 months	675 days	No injection-site tumors in any of the control or treated animals, and incidences of total tumors from other sites were comparable in all groups; survival was 32/50 in each group; primary report not available	Muller, 1968
	mouse/NMRI	20/NR	NR	0, 0.1, 1 or 10 mg suspended in aqueous gelatin once every 2 weeks for 20 weeks	22 months (life)	No skin or subcutaneous tumors in control or treated animals, and incidences of other tumors similar in all groups; injection site sarcomas developed in all mice similarly treated with benzo(a)pyrene; primary report not available	Muller, 1968

*Numbers in treated and control (if used) groups unless specified otherwise

TABLE 5-2

Mutagenicity Testing of Benzo(ghi)perylene

Assay	Indicator Organism	Compound and/or Purity	Application	Concentration or Dose	Activating System	Response	Comments	Reference
Reverse mutation	Salmonella typhimurium TA100	≥99%	plate incorporation	1-1000 µg/plate	with and without Aroclor 1254-induced rat liver S-9	mutagenic with S-9	Linear dose-related response from 100 µg/plate	Andrews et al., 1978
	S. typhimurium TA1537, TA1538	NR	plate incorporation	2-20 µg/plate	with and without Aroclor 1254-induced rat liver S-9	mutagenic in both strains with S-9	Dose-related response from ~2 µg/plate	Mossanda et al., 1979
	S. typhimurium TA98, TA100, TA1538	NR	fluctuation test	1 or 10 µg/mL	none	mutagenic at high dose in TA98 and TA1538	None	Mossanda et al., 1979
	S. typhimurium TA98	NR	plate incorporation	0.1-1000 µg/plate	with and without Aroclor 1254-induced rat liver S-9	weakly mutagenic with S-9	Also tested in TA100, TA1535, TA1537 and TA1538 but results were not reported	Salamone et al., 1979
Forward mutation	S. typhimurium TM677	NR	liquid suspension	NR	Aroclor or phenobarbital induced rat S-9	mutagenic	Range of doses not specified, 72 µM lowest positive concentration	Kaden et al., 1979
Cell transformation	hamster embryo cells	NR	intraperitoneal injection of pregnant hamsters	30 mg/kg	none	negative	None	Quarles et al., 1979

NR = Not reported

5.3. TERATOGENICITY

Weaver and Gibson (1979) exposed pregnant rats to graded airborne concentrations of uncharacterized oil shale containing PAH, including benzo-(ghi)perylene, on days 6-15 of gestation. No treatment-related teratogenic effects were observed during examination of fetuses obtained by Caesarean section on day 20 of gestation. Determination of a benzo(ghi)perylene specific effect is precluded, however, by the uncharacterized nature of the test material.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of benzo(ghi)-perylene could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding chronic and subchronic toxicity of benzo(ghi)-perylene could not be located in the available literature as cited in the Appendix.

5.6. OTHER RELEVANT INFORMATION

Other relevant information regarding the toxicity of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

5.7. SUMMARY

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo(ghi)-perylene in beeswax-trioctanoin mixture into groups of 34 or 35 rats produced a low incidence (4/34) of lung squamous cell carcinomas in the high-dose group after lifetime observation (Deutsch-Wenzel et al., 1983). Lung tumors did not occur in untreated or vehicle treated controls, but IARC (1983) indicated that the tumors in the treated group may be attributable to impurities in the test product, which was 98.5% pure.

Tumorigenicity of benzo(ghi)perylene was not demonstrated in mice in skin application studies involving up to 3 times/week treatment for 12 months (Lijinsky and Saffiotti, 1965; Van Duuren et al., 1970; Van Duuren and Goldschmidt, 1976; Muller, 1968; Hoffmann and Wynder, 1966); in skin initiation-promotion studies using croton oil as a promoter (Hoffman and Wynder, 1966) and PMA as a promoter (Van Duuren et al., 1970); and by bimonthly subcutaneous injections for 5 or 6 months (Muller, 1968). Enhancement of the dermal carcinogenicity of benzo(a)pyrene by simultaneous application of benzo(ghi)perylene has been reported in mice (Goldschmidt et al., 1973; Van Duuren et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of benzo(ghi)perylene.

Benzo(ghi)perylene was mutagenic to S. typhimurium strains TA98, TA100, TA1537 and TA1538 (Andrews et al., 1978; Mossanda et al., 1979; Salamone et al., 1979) and TM677 (Kaden et al., 1979) when assayed in the presence of exogenous metabolic activation preparations. Intraperitoneal injection of benzo(ghi)perylene into hamsters on day 10 of gestation did not induce transformations in the embryo cells (Quarles et al., 1979).

Pertinent data regarding toxic, teratogenic or other reproductive effects of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix.

6. AQUATIC TOXICITY

6.1. ACUTE

Pertinent data regarding the acute toxicity of benzo(ghi)perylene to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.2. CHRONIC

Pertinent data regarding the chronic toxicity of benzo(ghi)perylene to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.3. PLANTS

Pertinent data regarding the effects of benzo(ghi)perylene on aquatic plants could not be located in the available literature as cited in the Appendix.

6.4. RESIDUES

The only information regarding benzo(ghi)perylene and aquatic organisms consisted of bioconcentration and residue monitoring data. Obana et al. (1983) reported BCF values of 1000-1400 for short-necked clams, Tapes japonica, exposed to 0.06-0.13 µg/l for 1-7 days. Tsuji et al. (1985) found that clams (unspecified species) collected in Japanese waters contained benzo(ghi)perylene residues at concentrations of 0.23-0.95 µg/kg. Knutzen and Sortland (1982) reported the following levels (µg/kg dry weight) in species collected from polluted areas in Norway: mussel, Mytelis edulis, trace-87; periwinkle, Littorina littorea, 0.5; limpet, Patella vulgata, not detected; sponge, Halichondria panicea, 23; toothed wrack, Fucus serratus, 79; Ceramium rubrum, 4. Maccubbin et al. (1985) found that the stomach contents of white suckers, Catostomus commersoni, from eastern Lake Erie contained 2-14 ng/g benzo(ghi)perylene. Malins et al. (1985)

reported that stomach contents of English sole, Parophrys vetulus, collected in Puget Sound contained 75 ng/g dry weight. Dunn and Fee (1979) found that lobsters, Homarus americanus, caught in waters off eastern Canada contained benzo(ghi)perylene at an average concentration of 0.19 ng/g; however, it is a common practice for lobsters to be kept in impoundments for weeks or months before being sent to market. Frequently, these impoundments are made of creosote-treated wood, which would be a source of benzo(ghi)perylene and other PAH. Dunn and Fee (1979) found that benzo(ghi)perylene residues of impounded lobsters averaged 51 ng/g, a 250-fold increase over freshly caught lobsters. This may constitute a possible source of human exposure to benzo-(ghi)perylene.

6.5. SUMMARY

Pertinent data regarding the toxicity of benzo(ghi)perylene to aquatic organisms could not be located in the available literature as cited in the Appendix. Several studies reported data concerning residues in aquatic organisms. The following data were reported for species that are commonly eaten by humans: mussels from Norway, trace-87 ng/g (Knutzen and Sortland, 1982); clams from Japan, 0.23-0.95 ng/g (Tsuji et al., 1985); lobsters from eastern Canada, 0.19 ng/g before impoundment and 51 ng/g after impoundment (Dunn and Fee, 1979). Obana et al. (1983) reported BCF values of 1000-1400 for short-necked clams, T. japonica, exposed to 0.06-0.13 µg/ℓ for 1-7 days.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Exposure criteria and TLVs have been developed for PAH as a class, as well as for several individual PAH. OSHA has set an 8-hour TWA concentration limit of 0.2 mg/m³ for the benzene-soluble fraction of coal tar pitch volatiles (anthracene, benzo(a)pyrene, phenanthrene, acridine, chrysene, pyrene) (OSHA, 1985). NIOSH (1977) recommended a concentration limit for coal tar, coal tar pitch, creosote and mixtures of these substances of 0.1 mg/m³ of the cyclohexane-extractable fraction of the sample, determined as a 10-hour TWA. NIOSH (1977) concluded that these specific coal tar products, as well as coke oven emissions, are carcinogenic and can increase the risk of lung and skin cancer in workers. NIOSH (1977) also recommended a ceiling limit for exposure to asphalt fumes of 5 mg airborne particulates/m³ of air.

Environmental quality criteria, which specify concentration limits intended to protect humans against adverse health effects, have been recommended for PAH in ambient water. U.S. EPA (1980) recommended a concentration limit of 28 ng/ℓ for the sum of all carcinogenic PAH in ambient water. This value is based on a mathematical extrapolation of the results from studies with mice treated orally with benzo(a)pyrene and acknowledges the conservative assumption that all carcinogenic PAH are equal in potency to benzo(a)pyrene. On the basis of the animal bioassay data, daily consumption of water containing 28 ng/ℓ of carcinogenic PAH over an entire lifetime is estimated to keep the lifetime risk of cancer development <1 chance in 100,000.

U.S. EPA has not recommended an ambient water quality criterion for noncarcinogenic PAH as a class. U.S. EPA (1980) acknowledged that data suitable for quantitative risk assessment of noncarcinogenic PAH are essentially nonexistent.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the effects of benzo(ghi)perylene, in particular, could not be located in the available literature as cited in the Appendix; however, U.S. EPA (1980) noted that acute toxicity to saltwater aquatic life occurred at concentrations as low as 300 µg/ℓ of PAH in general, and would occur at lower concentrations in species more sensitive than those tested. U.S. EPA (1980) concluded that the database at that time was inadequate to generalize about chronic or acute toxicity to freshwater organisms.

8. RISK ASSESSMENT

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo(ghi)-perylene in beeswax-trioctanoin mixture into groups of 34 or 35 rats produced a low incidence (4/34) of lung squamous cell carcinomas in the high-dose group after lifetime observation (Deutsch-Wenzel et al., 1983). Lung tumors did not occur in untreated or vehicle treated controls, but IARC (1983) indicated that the tumors in the treated group may be attributable to impurities in the test product, which was 98.5% pure.

Tumorigenicity of benzo(ghi)perylene was not demonstrated in mice in skin application studies involving up to 3 times/week treatment for 12 months (Lijinsky and Saffiotti, 1965; Van Duuren et al., 1970; Van Duuren and Goldschmidt, 1976; Muller, 1968; Hoffmann and Wynder, 1966); in skin initiation-promotion studies using croton oil as a promoter (Hoffman and Wynder, 1966) and PMA as a promoter (Van Duuren et al., 1970); or by bimonthly subcutaneous injections for 5 or 6 months (Muller, 1968) (see Table 5-1). Enhancement of the dermal carcinogenicity of benzo(a)pyrene by simultaneous application of benzo(ghi)perylene has been reported in mice (Goldschmidt et al., 1973; Van Duuren et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of benzo(ghi)perylene.

Benzo(ghi)perylene was mutagenic to S. typhimurium strains TA98, TA100, TA1537 and TA1538 (Andrews et al., 1978; Mossanda et al., 1979; Salamone et al., 1979) and TM677 (Kaden et al., 1979) when assayed in the presence of exogenous metabolic activation preparations. Intraperitoneal injection of benzo(ghi)perylene into hamsters on day 10 of gestation did not induce transformations in the embryo cells (Quarles et al., 1979)

Although some lung tumors occurred in the intrapulmonary injection study with rats (Deutsch-Wenzel et al., 1983), possible cocarcinogenicity with benzo(a)pyrene on mouse skin occurred, and mutagenicity in different strains of S. typhimurium was demonstrated, the available data are inadequate for evaluation of carcinogenicity of benzo(ghi)perylene. The intrapulmonary injection study is limited by the single dose treatment schedule, unnatural route of respiratory exposure, possibility of impurities in the test compound and lack of corroborating data. The mutagenicity data are limited by a lack of data in other test systems. A cocarcinogen cannot be regarded as a complete or procarcinogen in the absence of supporting evidence. Evaluation of the carcinogenicity of benzo(ghi)perylene is further limited by the lack of studies employing oral or inhalation routes of exposure.

Pertinent data regarding toxic, teratogenic or other reproductive effects of benzo(ghi)perylene could not be located in the available literature as cited in the Appendix. This lack of data precludes calculation of an RfD (formerly called ADI) for benzo(ghi)perylene.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Pertinent data regarding the toxic or reproductive effects of benzo-(ghi)perylene could not be located in the available literature as cited in the Appendix. Weaver and Gibson (1979) exposed pregnant rats to graded airborne concentrations of uncharacterized oil shale containing PAH, including benzo(ghi)perylene, on days 6-15 of gestation. No treatment-related teratogenic effects were observed during examination of fetuses obtained by Caesarean section on day 20 of gestation. Calculation of an RQ from this study is precluded (Table 9-1) by the uncharacterized nature of the test material (U.S. EPA, 1983).

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Single intrapulmonary injections of 0.16, 0.83 or 4.15 mg benzo(ghi)-perylene in beeswax-trioctanoin mixture into groups of 34 or 35 rats produced a low incidence (4/34) of lung squamous cell carcinomas in the high-dose group after lifetime observation (Deutsch-Wenzel et al., 1983). Lung tumors did not occur in untreated or vehicle treated controls, but IARC (1983) indicated that the tumors in the treated group may be attributable to impurities in the test product, which was 98.5% pure.

Tumorigenicity of benzo(ghi)perylene was not demonstrated in mice in skin application studies involving treatment up to 3 times/week for 12 months (Lijinsky and Saffiotti, 1965; Van Duuren et al., 1970; Van Duuren and Goldschmidt, 1976; Muller, 1968; Hoffmann and Wynder, 1966); in skin initiation-promotion studies using croton oil as a promoter (Hoffman and Wynder, 1966) and PMA as a promoter (Van Duuren et al., 1970); or by bimonthly subcutaneous injections for 5 or 6 months (Muller, 1968) (see Table 5-1). Enhancement of the dermal carcinogenicity of benzo(a)pyrene by

TABLE 9-1

Benzo(ghi)perylene

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:

Dose:

Effect:

Reference:

RV_d:

RV_e:

Composite Score:

RQ: Data are not sufficient to derive an RQ.

simultaneous application of benzo(ghi)perylene has been reported in mice (Goldschmidt et al., 1973; Van Duuren et al., 1973; Van Duuren and Goldschmidt, 1976), indicating possible cocarcinogenicity of benzo(ghi)perylene.

Benzo(ghi)perylene was mutagenic to S. typhimurium strains TA98, TA100, TA1537 and TA1538 (Andrews et al., 1978; Mossanda et al., 1979; Salamone et al., 1979) and TM677 (Kaden et al., 1979) when assayed in the presence of exogenous metabolic activation preparations. Intraperitoneal injection of benzo(ghi)perylene into hamsters on day 10 of gestation did not induce transformations in the embryo cells (Quarles et al., 1979)

Although some lung tumors occurred in the intrapulmonary injection study with rats (Deutsch-Wenzel et al., 1983), possible cocarcinogenicity with benzo(ghi)perylene on mouse skin occurred, and mutagenicity in different strains of S. typhimurium was demonstrated, the available data are inadequate for evaluation of the carcinogenicity of benzo(ghi)perylene. The intrapulmonary injection study is limited oy the single dose treatment schedule, unnatural route of respiratory exposure, possibility of impurities in the test compound and lack of corroborating data. The mutagenicity data are limited by a lack of data in other test systems. A cocarcinogen cannot be regarded as a complete or procarcinogen in the absence of supporting evidence. Evaluation of the carcinogenicity of benzo(ghi)perylene is further limited by the lack of studies employing oral or inhalation routes of exposure.

IARC (1983) reported that there was insufficient evidence regarding the carcinogenic risk to humans and experimental animals associated with oral or inhalation exposure to benzo(ghi)perylene. Applying the criteria for evaluation of the overall weight of evidence for the carcinogenic potential for humans proposed by the Carcinogen Assessment Group of the U.S. EPA (U.S.

EPA, 1984), benzo(ghi)perylene is most appropriately designated a Group D, Not Classified chemical. Direct hazard ranking of benzo(ghi)perylene under CERCLA is therefore not possible.

10. REFERENCES

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APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

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Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

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